Amendments to the Specification:

Please replace paragraph **[00018]** with the following amended paragraph:

[00018] The inventors thus proceeded to characterize the genes and sequences in the 7 Mb region. It was discovered that for each of the genes present, the alleles of the genes carried by the most severely diabetic mice was the same as the alleles of the genes carried by the less severely affected mice, with the sole exception of the allele of the SorCS1 gene. Fig. 1 illustrates a genetic map of the genetic elements found in the 7 Mb region associated with the genetic difference. The region between map units 55 and 48 carried the genetic difference. The alleles of the SorCS3 gene turned out to be identical in the two strains of mice. As illustrated in Table 1 below, however, the susceptible mice had an allele of the SorCS1 gene that is three nucleotides different from that of the less severely diabetic mice. The resulting protein is also three amino acids different. This difference results in a genetic susceptibility to type 2 diabetes.

TABLE 1

SorCS1 mutations altering amino acids

Nucleotide			Amino Acid			
position in cDNA	B6	BTBR	position in protein	B6	BTBR	isoform(s)
172	C	T	~~50~~ 52	Thr	Ile	a,b,c
3433	C	T	1139	Ser	Phe	a
3462	T	C	1149	Ser	Pro	c

Please replace paragraph **[00019]** with the following amended paragraph:

[00019] The genomic and cDNA sequences of human SorCS-1 is known. The human SorCS-1 cDNA sequence (GenBank Accession No. NM_052918) and SorCS-1 amino acid sequence (GenBank Accession No. NP_443150) are incorporated herein as SEQ ID NO.~~1-2~~ 3-4, respectively. Also, shown in the sequence attachment hereto are the amino acid sequences of mouse (SorCS1a, SorCS1b, SorCS1c) and human (SorCS1a, SorCS1b, and SorCS1c). Also shown in Fig. 2 is an amino acid sequence alignment between mouse

SorCS1b (mSorCS1b) and human SorCS1 (hSorcs1). Note that the sequences are highly homologous, in fact have a sequence identity of 93%. It is this degree of identity that provides the ~~rational~~ rationale for the prediction that the genetic evidence from the congenic mouse model presented here does, in fact, predict the same genetic phenomenon in humans.

Please replace paragraph **[00020]** with the following amended paragraph:

[00020] From a diagnostic perspective, individual human beings can be examined for the allele of their SorCS-1 gene as a step in determining whether they are susceptible to developing type 2 diabetes. For example, the SorCS-1 cDNA sequence of an individual can be determined and the deducted amino acid sequence can be compared to SEQ ID NO:~~2~~ 4. If a mutation at the amino acid level is detected, especially if the mutation is one other than a conservative substitution, the individual can be identified as susceptible to developing type 2 diabetes.

Please replace paragraph **[00021]** with the following amended paragraph:

[00021] We have also discovered a similar allelic difference associated with susceptibility to diabetes, in a highly related gene. We have detected a C-to-A mutation in the SorCS 3 gene which is found in a Bedouin Arab family in which type 2 diabetes has a high occurrence rate. The mutation results in a Serine to Arginine mutation at amino acid 790 of the human SorCS3 amino acid sequence (SEQ ID NO:~~4~~ 2). While it is thus known that this mutation can permit the development of type 2 diabetes, there are certainly other mutations of these genes which cause the same susceptibility. The discovery of this mutation lends support to the concept that both of the related genes SorCS1 and SorCS3 can be the source of genetic susceptibility to type 2 diabetes.